RESULTS OF PERIPHERAL LASER PHOTOCOAGULATION IN PARS PLANITIS*

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ABSTRACT

Purpose: To determine the effect of peripheral retinal laser photocoagnulation (PLP) on visual acuity, intraocular inflammation, and other ocular findings, including retinal neovascularization in eyes with pars planitis.

Methods: A retrospective chart review of eyes with pars planitis that had undergone PLP.

Results: Twenty-two eyes in 17 patients with pars planitis had undergone treatment with PLP at 2 centers. The mean age at the time of treatment was 19.3 years. Following treatment, mean follow-up was 16.3 months (range, 6 to 37 months). Mean visual acuity was 20/60 preoperatively and 20/50 postoperatively. This level of improvement was not statistically significant ($P > .10$), but there was a statistically significant decrease in the use of corticosteroids between the preoperative examination and the last postoperative examination (86% versus 27%, $P < .05$). There was also a statistically significant decrease in vitritis at the last follow-up ($P = .0008$) and a decrease in neovascularization of the vitreous base ($P = .03$) and in clinically apparent cystoid macular edema ($P = .02$). Epiretinal membranes were noted in 23% of eyes preoperatively and in 45% of eyes postoperatively. Only one of these epiretinal membranes was considered to be visually significant. One eye developed a tonic dilated pupil, which slowly improved.

Conclusions: Although the long-term natural history of clinical findings in pars planitis is not well documented, PLP appears to decrease the

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need for corticosteroids while stabilizing visual acuity. It also appears to decrease vitreous inflammation. PLP has few complications and should be considered in patients with pars planitis who are unresponsive or have adverse reactions to corticosteroids.

INTRODUCTION

Pars planitis is an idiopathic intermediate uveitis that usually affects persons in the second to third decades of life. The course of this disease is variable and ranges from a self-limited disease to a severe course.[1] In cases of decreased vision or severe floaters, treatment has consisted of topical, periocular, or systemic corticosteroids.[2,3] Aaberg and associates[4] reported improvement in vision and decreased vitritis with the use of peripheral retinal cryoablation. Others have subsequently reported similar findings, although there have been reports of retinal detachment following the use of peripheral retinal cryoablation, and this limits its use.[5-7]

Recently, Park and colleagues[8] described the use of peripheral laser photocoagulation in 10 eyes with pars planitis and neovascularization of the vitreous base (NVVB). They showed that the NVVB showed signs of regression. In addition, there was an impression that the vitritis decreased after photocoagulation. The follow-up was limited, and the number of cases was small. We now present long-term follow-up on the original 10 eyes and data from 12 additional eyes with pars planitis.

METHODS

All patients were seen, diagnosed, and treated either by the vitreoretinal service of the Department of Ophthalmology at the Medical College of Wisconsin, Milwaukee, or at Northwestern University, Chicago. The Snellen visual acuity of the treated patients with pars planitis who underwent peripheral laser photocoagulation was converted into logmar for statistical analysis. In addition, vitreous inflammation was graded preoperatively and at the last postoperative visit using the grading scheme of the American Uveitis Society.[9] Other data included the presence or absence of clinically observed cystoid macular edema, the use of vitrectomy, and the presence of NVVB. The use and route of corticosteroid therapy preoperatively and at the latest postoperative visit were also recorded. The statistical analysis was performed using the SAS statistical analysis package (Statistical Analysis System). Paired *t* tests were used to compare the recorded data preoperatively and at the last examination. The preoperative and postoperative use of corticosteroids and development of epiretinal membranes were compared by using the McNemar test for paired comparisons. A *P* value less than or equal to .05 was considered statistical-

ly significant.

Laser photocoagulation was performed using an indirect ophthalmoscopic delivery system in 21 eyes. The argon or diode infrared laser was used. Photocoagulation burns were placed confluently in 3 or 4 rows just posterior to the snowbank. Treatment was extended to the one-o'clock hour superior to the snowbank on each side. The burn intensity was titrated to a white-gray burn. In 1 eye (No. 9 OS), laser photocoagulation was performed using a slit-lamp delivery system in a pattern similar to that made with the indirect ophthalmoscopic delivery system as well as to areas of inferior phlebitis.

RESULTS

A total of 22 eyes in 17 patients were treated (Table I). Fourteen patients were female and 3 were male. The mean age was 19.3 years (range, 6 to 41 years). The mean follow-up was 16.3 months (range, 6 to 37 months).

Six eyes underwent pars plana vitrectomy concomitantly with the peripheral laser photocoagulation.[2] Three eyes had mild vitreous hemorrhages preoperatively. One of these had a preoperative visual acuity of 20/30, and the other 2 had visual acuities of 20/25.

The mean pretreatment visual acuity of all eyes was 20/60 (logmar 0.515), and the mean posttreatment acuity was 20/50 (logmar 0.445). This line of improvement was not statistically significant ($P > .1$). The statistical analysis was also performed excluding those who had also undergone a vitrectomy or had a preoperative vitreous hemorrhage. Excluding these cases, the mean preoperative visual acuity was 20/65, and the final mean visual acuity was 20/55. Again, this was not statistically significant ($P > .1$). Regression analysis showed that lower age ($P = .022$) and better initial visual acuity ($P = .0004$) were associated with improvement following peripheral laser photocoagulation.

The amount of vitritis decreased following peripheral laser photocoagulation (PLP). The pretreatment mean vitreous cell grade was 2.3, and the posttreatment mean cell grade was 0.87 ($P = .0008$). Excluding eyes that underwent a concomitant vitrectomy, pretreatment cell grade was 2.2; at the final postoperative examination it was 0.87 ($P = .013$). Excluding cases with preoperative vitreous hemorrhage or a vitrectomy, there was still a significant improvement in vitritis, from a grade of 2.4 to 0.87 ($P = .02$). There was a trend toward improvement with longer postoperative follow-up time, although this did not reach statistical significance ($P = .06$).

Neovascularization of the retina was seen preoperatively in 12 eyes (55%). Eleven had only NVVB, and 1 had neovascularization of the disc as well. Postoperatively, 2 eyes (9%) had clinically visualized NVVB ($P = .012$). Preoperatively, clinically visualized cystoid macular edema was

noted in 12 eyes (55%), and at the last follow-up, cystoid macular edema was noted in 5 eyes (22%). There was a statistically significant decrease in cystoid macular edema in eyes that underwent vitrectomy and PLP and those that only underwent PLP ($P = .005$ and $P = .02$, respectively).

There was less corticosteroid use at the final postoperative follow-up examination than prior to PLP. Corticosteroids were used in 19 eyes (86%) preoperatively and in 6 eyes (27%) at the last examination ($P < .05$).

Topical corticosteroids were used preoperatively in 7 eyes (32%), and because of a glaucomatous response in 2 eyes and poor control in the other 5 eyes, PLP was performed. Postoperatively, 1 patient (2 eyes) required topical corticosteroids alone. Periocular corticosteroids alone or in combination with topical corticosteroids were used in 3 eyes (14%) preoperatively and in 3 eyes postoperatively. Systemic corticosteroids either alone or in combination with periocular or topical corticosteroids were used to treat 9 eyes of 9 patients (41%) preoperatively and for 1 eye of 1 patient (4.5%) postoperatively.

Epiretinal membranes were noted in 23% of eyes preoperatively and in 46% of eyes at the last follow-up. This was not a statistically significant difference ($P = .11$). Only 1 (4.5%) of these epiretinal membranes caused enough macular distortion to be considered visually disabling.

One eye (4.5%) developed a dilated pupil that did not accommodate. This eye had extensive snowbank formation and underwent 8 clock hours of PLP. Presumably, ciliary innervation to the pupil may have been affected. This complication was transient, and at the last follow-up the pupil had become reactive and was symmetric to the fellow untreated pupil. No retinal detachments or other complications occurred in any of the patients.

DISCUSSION

A previous study by Parks and associates[8] showed that PLP caused regression of NVVB. It also appeared to decrease inflammation, but only 10 eyes were treated, and this was an insufficient number of eyes to achieve statistical significance. There were no complications noted; therefore further evaluation of this technique for the treatment of inflammation in patients with pars planitis appeared warranted. In this retrospective study, longer follow-up of the 10 originally treated eyes is provided, along with the results of 12 more eyes with pars planitis that were treated with PLP.

Vision at the last postoperative examination was not significantly different from that at preoperative examination. It is important to note, however, that there was a statistically significant decrease in the requirement of corticosteroids following PLP to maintain vision. There was a concern that the apparent steroid-sparing effect of PLP might in fact be an effect of the concomitant vitrectomy in 6 eyes that had undergone the procedure

Table I: Results of Peripheral Laser Photocoagulation in Pars Planitis

Pt No. Age/Sex	Eye	Initial VA	Previous Steroid Tx	Initial Findings*	Tx	Final VA	Follow-Up	Final Findings*
1/12/F	OS	20/100	Topical	3+ cells, CME, NVVB	PPV, cryo, PLP	20/50	37 mo	0 cell
	OD	20/20	Topical	3+ cell, extrafoveal TRD, NVVB	PLP	20/20	14 mo	1+ cell, TRD
2/33/F	OS	20/50	Topical/ periocular	ERM, 2+ cell, CME, NVVB	PPV, PLP	20/100	34 mo	0 cell, NVVB, ERM
	OD	20/80	Topical/ periocular	ERM, 2+ cell, CME, NVVB	PLP	20/70	24 mo	0 cell, NVVB, ERM
3/15/M	OS	20/25	Topical/ periocular/ systemic	2+ cell, NVVB, CME, ERM	PLP	20/40	14 mo	1-2+ cell, CME, ERM
	OD	20/200	Topical/ periocular/ systemic	3+ cell, NVVB, CME, ERM	PLP	20/200	33 mo	1+ cell, 3+ PSC, ERM
4/29/F	OS	20/200	Topical/ systemic	2-3+ cell, NVVB, CME	PPV, PLP	20/50	23 mo	1+ PSC, 0 cells, CME
	OD	20/100	Topical/ systemic	2-3+ cell, NVVB, CME	PPV, PLP	20/200	13 mo	1+ PSC, 0 cells, CME
5/9/F	OD	20/30	Topical	3+ cell, NVVB, Coats' response	PLP	20/20	20 mo	1+ cell, ERM

TABLE I (CONTINUED): RESULTS OF PERIPHERAL LASER PHOTOCOAGULATION IN PARS PLANITIS

PT NO. AGE/SEX	EYE	INITIAL VA	PREVIOUS STEROID TX	INITIAL FINDINGS*	TX	FINAL VA	FOLLOW-UP	FINAL FINDINGS*
6/12/M	OD	20/25	Topical	2+ cell	PLP	20/15	17 mo	0 cell
7/15/F	OS	20/40	Topical	3+ cell	PLP	20/15	3 mo	no NVVB, rare cell
8/6/F	OS	20/50	Topical	1+ cell, NVVB	PLP	20/30	14 mo	2-3+ cell, ERM, topical steroids
	OD	20/25	Topical	2+ RBCS, 1+ cell, NVVB	PLP	20/50	11 mo	1-2+ cell, no vit heme, ERM, topical steroids
9/25/M	OS	20/25	None	trace cell, NVD with vit heme	PLP	20/20	27 mo	decreased NVD, no vit heme, ERM
10/26/F	OS	20/250	Periocular/ systemic	2+ cell, CME	PLP	20/160	4 mo	1+ cell, no CME
11/8/F	OD	3/200	Topical/ systemic	2+ cell, cataract, NVVB	PPV, PPLX, PLP	20/100	18 mo	0 cell
12/17/F	OD	20/50	Periocular/ systemic	2 cell, CME	PLP	20/30	7 mo	0 cell
13/25/F	OD	20/40	Periocular	3-4+ cell, CME	PLP	20/40	9 mo	trace cell, CME, ERM, periocular steroids

TABLE I (CONTINUED): RESULTS OF PERIPHERAL LASER PHOTOCOAGULATION IN PARS PLANITIS

PT NO. AGE/SEX	EYE	INITIAL VA	PREVIOUS STEROID TX	INITIAL FINDINGS*	TX	FINAL VA	FOLLOW-UP	FINAL FINDINGS*
14/26/F	OS	20/30	None	1-2+ cell, ERM, CME	PLP	20/63	9 mo	3+ cell, CME, ERM, periocular, systemic steroids
15/24/F	OS	20/20-3	None	2+ vit. cells, CME	PLP	20/30	11 mo	2+ cell, CME, periocular steroids
16/41/F	OS	20/40-	Periocular, systemic	1-2+ PSC, 3+ cell, CME	PPV, PLP	20/30	13 mo	1+ cell, 1-2+ PSC, ERM, CME, periocular steroids
17/13/F	OD	20/50-	Systemic	PSC, 2+ cell	PLP	20/25-	6 mo	PSC, trace-1+ cell

CME, cystoid macular edema; ERM, epiretinal membrane; NVD, neovascularization of disc; NVVB, neovascularization of vitreous base; PLP, peripheral retinal laser photocoagulation; PPLX, pars planitis lensectomy; PPV, pars plana vitrectomy; PSC, posterior subcapsular cataract; RBC, red blood cell, TRD, traction retinal detachment.

*Cell grade: 0, trace, 1+, 2+, 3+, and 4+

at the same time as the PLP. Exclusion these eyes from the statistical analysis still showed a similar statistically significant effect of PLP. Three eyes had a mild preoperative vitreous hemorrhage, and analysis of results excluding these eyes as well did not change the fact that preoperative vision and the vision at the last follow-up examination were not statistically different, even though there was a statistically significant decrease in the need for corticosteroids with maintenance of stable vision following PLP.

There was a statistically significant large improvement in the grade of cells in the vitreous after treatment. Even after excluding eyes that had preoperative vitreous hemorrhage or who also underwent vitrectomy, there was still a statistically significant improvement. Although there was less use of corticosteroids at the last follow-up examination, the amount of vitreous cells had decreased from greater than a grade of 2 to less than a grade of 1. There was a trend toward improvement with longer follow-up times. This may be explained by the fact that the vitreous cellular infiltration is slow to clear following PLP because of sequestration of the cells by the vitreous gel.

Confirming our original case series, the amount of NVVB improved as well.[8] Following the apparent improvement in inflammation in the original set of eyes that had NVVB, we expanded the use of PLP to eyes with inflammation that did not have clinically obvious NVVB. It may be that most eyes with snowbanks have NVVB by histopathologic examination, but only about 6.5% are noted to have it by clinical evaluation.[1,10] The discrepancy between the histologic and ophthalmoscopic determination of NVVB may be either that ophthalmoscopic determination of NVVB may be difficult in mild cases of pars planitis or that only the most severe cases of pars planitis have had histopathologic examination and that in these severe cases, NVVB is invariable. It appears that the clinical presence of NVVB does not appear to be important for the effect of PLP on inflammation in cases of pars planitis.

Although fluorescein angiograms were not performed in all cases, clinical evaluation of the presence of cystoid macular edema was performed and recorded. There was a statistically significant decrease in clinically apparent cystoid macular edema following PLP, although there could still be angiographic cystoid macular edema in these cases.

Corticosteroids are a mainstay of therapy in pars planitis. Complications of systemic steroid use are numerous. Since many patients with pars planitis are young, specific systemic complications that have to be considered include growth retardation, acne, and obesity. Periocular and topical corticosteroids do not cause the systemic complications, but they are associated with glaucoma, ptosis, and cataracts.[3,11] Because of the serious complications from corticosteroid use, its use in cases of pars planitis has been limited to patients with significant visual loss or with disabling floaters.

Some investigators[12,13] have used diathermy through a scleral flap in this disease. This was performed directly over the snowbanks The investigators noted that the snowbank decreased in size and the inflammation lessened as well. The problem with the technique is that it requires making scleral flaps.

Peripheral retinal cryotherapy has supplanted diathermy and has been used as an alternative to corticosteroids or in cases unresponsive to corticosteroids.[4] In their initial report, Aaberg and associates[4] showed that following treatment, 13 of 23 eyes (57%) had a decrease in vitritis. They also noted an improvement in visual acuity, although statistical analysis was not performed. Others[5-7] have subsequently reported an apparent treatment effect as well.

How peripheral diathermy or cryotherapy functions is unknown. Although cryotherapy involves direct treatment of the snowbank, the reports that have used this technique all describe treatment of the retina posterior to the snowbank in association with treatment of the snowbank.[4-7] Josephberg and colleagues[5] performed peripheral fluorescein angiography in eyes with pars planitis before and after peripheral retinal cryotherapy. Prior to cryotherapy there were areas of marked peripheral fluorescein leakage. After treatment these areas of leakage were not noted. Peripheral cryoablation may decrease the release of angiogenic factors elaborated by the ischemic peripheral retina in the areas of capillary dropout. These angiogenic factors may cause not only growth of neovascularization but also leakage from the normal retinal vasculature. One angiogenic factor, vascular endothelial growth factor, which is a putative angiogenic factor elaborated in some cases of retinal ischemia, is 50,000 times more potent than histamine in causing vascular leakage. Another possibility is that peripheral cryotherapy may directly ablate the NVVB.[4]

The fact that PLP appears to decrease inflammation similar to peripheral retinal cryoablation suggests that direct treatment of the snowbank may be unnecessary and that treatment of only the peripheral retina is sufficient. Since the investigators who have performed cryoablation treated both the snowbank and the peripheral retina, they could not determine the effect of treating just the peripheral retina.[4-7]

Treatment of the peripheral retina may be a safer approach to this disease. Indeed, in our series no retinal detachments occurred following peripheral laser photocoagulation, while peripheral retinal cryoablation has been associated with retinal detachments.[6,7] It could be that the direct treatment of the snowbank causes contraction of the vitreous base and secondary retinal tears. In one study all cases of retinal detachment occurred within the first 6 weeks following retinal cryotherapy (Wolf M and Mieler WF, American Academy of Ophthalmology, 1992). Additionally, there may be less breakdown of the blood-retinal barrier following laser photocoag-

ulation than with retinal cryoablation.[14]

A complication noted in our series was a tonic dilated pupil that slowly improved. This complication has been noted following laser panretinal photocoagulation in cases of diabetic retinopathy.[15] Probably photocoagulation near the horizontal periphery caused reversible damage to the underlying long posterior ciliary nerves.

Another possible complication is the development of epiretinal membranes following PLP. Epiretinal membranes may occur following laser retinopexy of retinal tears. The incidence in this situation is probably very small.[16] Epiretinal membranes are also seen in eyes with pars planitis that have not undergone PLP, as well as in eyes undergoing any form of cryotherapy. In a study by Malinowski and associates[1] of eyes with pars planitis that had not undergone PLP, the prevalence of epiretinal membranes was 35%. In their series, only 6.5% of eyes had epiretinal membranes that caused marked vascular tortuosity and straightening of vessels. In our present series, 23% of eyes had epiretinal membranes noted preoperatively and 46% of eyes had them at the last follow-up. This was not a statistically significant difference. Though the epiretinal membranes could have been caused by the PLP, they may also be a part of the natural history of the disease. Regardless, all but 1 eye (4.5%) had mild epiretinal membranes, which were not severe enough to cause visual impairment. This may be no worse than the follow-up results in pars planitis eyes that have not undergone PLP.[1]

The limitations of the present study were that it was retrospective and several cases underwent concomitant vitrectomy. We controlled for the latter concern in our statistical analysis. In addition, there is no good natural history study of patients with pars planitis, but in our experience these patients appear to be doing better than expected. Peripheral laser photocoagulation requires further evaluation, but considering the apparent lack of significant complications, this treatment appears to be a viable corticosteroid-sparing alternative in patients with pars planitis or may be used in place of and prior to the use of cryotherapy in patients unresponsive to corticosteroids.

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DISCUSSION

DR THOMAS W. GARDNER. Dr Pulido and colleagues have reported their findings on the effects of peripheral laser photocoagulation in 22 eyes of 17 patients with severe pars planitis. The strengths of this paper are multiple. The authors have extensive experience in the management of pars planitis and have studied patients from 2 major midwestern eye centers. They have carefully described the methods and the criteria by which the patients were treated and evaluated and have used multiple end points to evaluate the effects of peripheral laser photocoagulation. Moreover, the follow-up was extensive, with a mean of 16.3 months.

The most impressive improvements were in the reduction of vitritis, peripheral and optic disk neovascularization, cystoid macular edema, and requirement for steroids. These are important parameters because they have direct impact on the ocular morbidity and impairment of the patients. The lack of visual improvement is not surprising and may relate to the fact that the patients likely had chronic pars planitis with CME prior to being considered for laser treatment. The only reported side effect was

a transient mydriasis.

The relative weaknesses of this report are few and relate primarily to its retrospective nature, which the authors acknowledge. Although pars planitis may spontaneously improve, the temporal relationship between peripheral laser surgery and improvement suggests a causal relationship. Given the considerable complications of chronic periocular or systemic steroid therapy, the authors have provided further evidence for direct ablation of the peripheral retina and expanded our treatment options. I congratulate the authors on their careful study and encourage them to develop a larger, prospective trial.

I would like to ask Dr Pulido whether he feels that, on the basis of their data, peripheral laser photocoagulation should be reserved for patients with steroid-resistant pars planitis or whether laser treatment should be instituted earlier.

DAVID KNOX, MD. I would like to thank Dr Pulido for sending me a copy of his paper so that I had more data to study than was provided in the abstract.

The pars planitis syndrome and this report are examples of the difficulties in making science out of the care of patients with ocular inflammation.

The first difficulty is one of diagnosis and classification — not all ophthalmologists agree.

The second difficulty is in defining etiologies — our western world wants a single reason for a given syndrome — it is my opinion that most ocular inflammations are multifactoral.

The worse difficulty is therapy. Today much of the care of patients with ocular inflammations is in the hands of the retino vitreous specialists. Today's medical scientists hold as a gold standard, the single variable bench experiment. This report is an example of how difficult it is to meet that requirement. The patients in this retrospective report from two different centers had different medical and surgical treatments, both before and after laser treatments of their peripheral retinae.

I have spend a significant part of the last 35 years studying and thinking about pars planitis and believe that allergic mechanisms are important aspects of the pathophysiology. I like to think of this disorder as chronic ocular urticaria.

This fluorescein angiogram demonstrates what I believe is the core functional abnormality, diffuse leakage from retinal capillaries, not the accumulation of debris in the inferior fundus.

In this composite picture we can see leakage at the maculae of both eyes, the symptomatic left eye and the asymptomatic right eye.

After the patient stopped cigarette smoking for 2 months, the lower set

of angiograms show marked improvement in the leakage of capillaries in both eyes.

I wish that Dr Pulido would explain how a focal peripheral laser treatment can benefit the whole eye.

My final question asks whether there was an opportunity to treat only one eye of bilaterally but similarly involved eyes of one patient?

ALLEN KREIGER, MD. I have a couple of questions. First of all, you mentioned that your treatment was to the snowbanks just posterior to the vitreous base. Was the treatment just to the inferior half of the eye or was it over the whole 360 degrees? Did this have any affect on the visual field?

DOUGLAS JABS, MD. Dr Pulido was kind enough to send me a draft manuscript before the meeting. In previous work these authors have demonstrated that peripheral laser photocoagulation results in regression of neovascularization in patients with pars planitis, and they have reported that it appears to be safer than cryotherapy. Therefore, the procedure has value in patients with pars planitis.

The more problematic issue is its role in controlling inflammation in patients with pars planitis. In this manuscript, there is no control group, not even an historical one. Although a prospective, randomized, controlled clinical trial of laser photocoagulation would be ideal, the disease does not occur with sufficient frequency that any single center could conduct such a trial. However, a control group, either contemporaneous or historical, would provide some information for comparison purposes.

The authors' primary reason for suggesting that peripheral laser photocoagulation has efficacy in controlling the inflammation was the lower dose of steroids required at follow-up when compared to those at the time of laser photocoagulation. The problems with this analysis are that some patients' disease will "burn out" over time resulting in lower steroid needs, and that even those patients who require long-term steroid therapy will often require lower doses of steroids after careful tapering of the medication. Furthermore, most of the patients in this series whose inflammation improved also received steroid therapy, and for the few patients who did not receive concurrent steroid therapy, more often than not the inflammation was worse at follow-up. This result suggests that laser photocoagulation may be useful as an adjunct to corticosteroid therapy, rather than as primary therapy for the inflammation in pars planitis. As such, I believe that this report presents exciting preliminary data, but that we must be careful about recommending laser photocoagulation as primary therapy for ocular inflammation in patients with pars planitis.

PAUL TORNAMBE, MD. How many of the patients had macular edema?

How many had vitrectomy? In any of the cases, did the macular edema get worse after peripheral photocoagulation?

FRONCIE GUTMAN, MD. In my clinical experience with pars planitis, retinal vascular leakage causing macular edema develops as a late event due to chronic intraocular inflammation. Since many patients with pars planitis never develop macular edema, it seems inappropriate to consider peripheral laser treatment and/or retinal cryopexy prior to the development of vision threatening complications such as macular edema.

I have treated approximately 12 patients with peripheral retinal cryopexy who developed macular edema secondary to pars planitis. All of these cases had bilateral pars planitis and I elected to treat only one eye. Sixty percent of the treated eyes shows resolution of their macular edema and improvement in visual acuity.

ROBERT B. WELCH, MD. Interestingly enough it's been 38 years since I wrote the paper on pars planitis and it's still an enigma. Early on I received a lot of grief from some for my choice of the term but because it described a morphological entity and the name was easy to remember it has stuck. I agree that treating the peripheral retinal can improve the inflammation. I first treated the region of the ora serrata with diathermy under scleral flaps in 1959 and later used cryotherapy in the same area. In a number of cases there was lessening of inflammation and improvement of vision. Unfortunately the use of cryotherapy by some was excessive which led to retinal detachments. It was never clear to me why treating the region of the "snowbank" would improve the situation. Although we can correlate pars planitis occasionally with such entities as multiple sclerosis and sarcoidosis the etiology is usually illusive. I feel that the cause of the syndrome is often multifactorial and that there are a number of variants such as those with peripheral pseudoangiomas. Since the "snowbanks" are usually below they have been thought to be gravitational but this would not explain those cases with 360 degree involvement. I feel we still have much to learn about pars planitis.

W. RICHARD GREEN, MD. Dr Kenneth Kenyon and associates studied several snowbanks of pars planitis and found them to consist of collapsed and condensed vitreous, to which retinal vessels and retinal astrocytes contribute. New collagen is produced by the astrocytes. Minor hyperplasia of the ciliary epithelia also contribute. That is the description of what we have more recently called "anterior proliferative vitreoretinopathy. I do not see how treating the snowbank affects the overall outcome. Such trials are valuable and sometimes give clues to the nature of the disease.

JOSE PULIDO, MD. I knew this was going to be interesting because even the name is controversial. As far as the term goes, at the last America Uveitis Society winter meeting in 1997, we decided we could call this form of intermediate uveitis, pars planitis.

In young patients, oral steroids cause a large number of problems, not just the problems seen in adults including changed mentation and osteoporosis but also growth abnormalities, obesity, and acne. I therefore think that in cases that are not responsive to periocular steroids or in patients that develop a glaucomatous response to ocular steroids, peripheral laser photocoagulation (PLP) should be tried and especially before the use of any systemic immunosuppresive agents. Peripheral cryotherapy has been used as well but it has a risk of retinal detachments in these patients and this has not been seen with PLP.

Dr. Knox has studied this disease and its possible allergic associations but there have been no good peer-reviewed papers on this relationship and we are still awaiting them. The only systemic association of this disease has been associated by us, and that association is with multiple sclerosis. This is because of a similar HLA predisposition, namely HLA-DR2.

Dr. Knox also wanted to know about the follow-up on fellow eyes. Some of the untreated fellow eyes eventually did require PLP as well.

Dr. Krieger asked if PLP caused visual field defects. We placed 3 or 4 rows of laser photocoagulation in the retina just posterior to the snowbank. This often involved the inferior half of the periphery. This treatment was in the far periphery so if it did affect the visual fields, it would be a very peripheral superior scotoma. By treating the areas posterior to the snowbanking, we seem to have avoided the problems of retinal detachments sometimes associated with cryotherapy.

At the time of the surgery, we gave a subconjunctival injection of celestone but ultimately the patient required less steroids and the vitritis decreased so we think the treatment was effective. We did not have a control group and this was a retrospective study, as we mentioned. However, this was the first study of a surgical intervention in pars planitis that did try to determine if there was a statistically significant outcome.

Finally, a previous study by Dr. Green and a study by Dr. Rao do show the presence of neovascularization at the level of the vitreous base. We are attempting to stop growth of the neovascular tissue by laser photocoagulation.